Brief Report
Volume 7:1, 2022

Journal of
Molecular Histology and Medical Physiology

Open Access

Signalling by osteocyte cells in relation to bone

Jenneke Klein*

Department of Morphology and Cellular Biology, Faculty of Medicine, Oviedo University, Oviedo, Spain

Brief Report

In the last few decades, our understanding of bone biology has shifted dramatically. Osteocytes are multifunctional bone cells surrounded by calcified bone matrix, and they were thought to be largely dormant cells for decades. However, it is now recognised that osteocytes are highly active cells that are required for the correct function of the skeleton, and that they play key roles in a variety of physiological processes both within and beyond the bone microenvironment. The present level of knowledge of the osteocyte is highlighted and updated in this review, which emphasises on its roles in bone remodelling and mineral homeostasis, as well as its recently revealed endocrine function. Sclerostin (a protein that acts as a negative regulator of bone mass) and FGF-23, the most important osteocyte-released endocrine factor that regulates phosphate metabolism, are both secreted by osteoclasts. Furthermore, osteocytes have the ability to act as mechanosensory cells, converting mechanical strain into chemical signals for effector cells (osteoblasts and osteoclasts). Because it regulates both osteoblast and osteoclast activity, the osteocyte plays a vital role in bone biology, particularly in the remodelling process. Finally, the study examines how bone biology is used in clinical practise, as well as innovative treatments for bone loss illnesses.

The 'osteoimmune' system is made up of bone tissue, which serves as a locomotor organ, mineral reservoir, and primary lymphoid organ for the maintenance of haematopoietic stem cells. Under pathological situations, abnormal and/or extended immune responses disrupt bone and mineral metabolism, which is maintained by the balanced action of bone cells such as osteoclasts, osteoblasts, and osteocytes. However, osteoimmune interactions are not limited to the immune system's unidirectional effect on bone metabolism. The function of osteoprogenitor cells in haematopoietic stem cell regulation and osteoblast-mediated suppression of haematopoietic malignancies have both been discovered in recent years as impacts of bone cells on immune regulation. Furthermore, the dynamic reciprocal interactions between bone and malignancies in distant organs have piqued interest, broadening the scope of osteoimmunology. We examine new ideas in the osteoimmune debate in health and illness in this article.

The regulated balance between bone cell populations, particularly bone-forming osteoblasts, bone-resorbing osteoclasts, and the osteocyte, the mechanosensory cell type, results in the process of bone remodelling. The main cells involved in bone resorption are osteoclasts, which are generated from the hematopoietic stem cell lineage. The equilibrium is lost in osteolytic illnesses such rheumatoid arthritis, periodontitis, and osteoporosis, and it shifts in favour of bone resorption. Understanding the mechanics of osteoclast development and bone resorption is critical. Osteocytes have been found to express Receptor activator of nuclear factor B ligand (RANKL), an important factor in the development of osteoclasts. The most critical component for physiologically supported osteoclast development in the developing skeleton and pathological bone resorption, such as experimental periodontal bone loss, is RANKL released by osteocytes. TNF- stimulates osteoclast development by increasing RANKL expression in osteocytes. TNF- also stimulates the production of osteoclasts via increasing sclerostin expression in osteocytes.

These findings imply that osteocyte-related cytokines directly promote the development of osteoclasts and bone resorption. The osteocyte as master regulator of bone resorption and effector in osteoclast production is discussed in this study, which summarises the most recent findings on bone resorption-related cytokines. In humans, osteoocytes are the most common (95 percent) and have the longest half-life (25 years). Because they are buried inside the thick bone matrix, osteocytes had previously been thought of being vestigial cells in bone. However, during the last 30 years, it has become obvious that osteocytes are just as crucial in maintaining bone homeostasis as bone building osteoblasts and bone resorbing osteoclasts. In a complex lacuno-canalicular system, the osteocyte cell body and dendritic processes dwell in bone, allowing direct networking of osteocytes to their surrounding osteocytes, osteoblasts, osteoclasts, bone marrow, blood vessels, and nerves. The applied mechanical stress on bone is translated into cellular communication and bone adaption regulated via osteocyte mechanosensing.

The osteocyte lacuno-canalicular system is very effective in transferring external mechanical force from the bone to the cell body and dendritic processes of the osteocyte via fluid displacement in the lacuno-canalicular gap. Osteocyte mechanotransduction maintains bone homeostasis by regulating the development and function of osteoblasts and osteoclasts. Sclerostin, cathepsin K, Wnts, DKK1, DMP1, IGF1, and RANKL/OPG are among the proteins and signalling molecules produced by osteoocytes that modulate osteoblast and osteoclast activity. Sclerosteosis, van Buchem disease, hypophosphatemic rickets, and WNT1 and plastin-3 mutation-related disorders are just a few of the rare bone diseases linked to disturbed osteocyte functioning. Over the last 15 years, meticulous research on disturbed osteocyte function in rare bone illnesses has led to the discovery of a number of innovative treatment medicines for bone diseases. The role of sclerostin in bone homeostasis was discovered through genetic, molecular, and cellular studies of sclerosteosis and van Buchem disease, leading to the development of a sclerostin antibody to treat osteoporosis and other bone degenerative illnesses. The mechanism of many other rare bone diseases, as well as the involvement of the osteocyte in their development, are yet unknown. what we've learned about the role of the osteocyte in uncommon bone diseases during the previous 30 years. Future research directions for developing novel therapeutic medicines targeting osteocyte activities to treat both common and unusual bone ailments [1-5].

***Address for Correspondence:** Jenneke Klein, Department of Morphology and Cellular Biology, Faculty of Medicine, Oviedo University, Oviedo, Spain, E-mail: Jennekek@um.es*

Received: 06 January, 2022, Manuscript No. jmhmp-22-54013; **Editor assigned:** 07 January, 2022, PreQC No. P-53894; **Reviewed:** 11 January, 2022, QC No. Q-53894; **Revised:** 16 January, 2022, Manuscript No. R-53894; **Published:** 21 January, 2022, DOI: 10.37421/mhmp.2022.7.30

References

1. Yan, Yongyong, Liping Wang, Linhu Ge, and Janak L. Pathak. "Osteocyte-mediated translation of mechanical stimuli to cellular signaling and its role in bone and non-bone-related clinical complications." Curr Osteoporos Rep 18 (2020): 67-80.
2. Pathak, Janak L., Nathalie Bravenboer, and Jenneke Klein-Nulend. "The osteocyte as the new discovery of therapeutic options in rare bone diseases." Front Endocrinol 11 (2020): 405.
3. Paul, Graeme R., Angad Malhotra, and Ralph Müller. "Mechanical stimuli in the local in vivo environment in bone: computational approaches linking organ-scale loads to cellular signals." Curr Osteoporos. Rep 16 (2018): 395-403.

4. Lerner, Ulf H., Elin Kindstedt, and Pernilla Lundberg. "The critical interplay between bone resorbing and bone forming cells." J Clin Periodontol 46 (2019): 33-51.

5. Curtis, Kimberly J., Thomas R. Coughlin, Devon E. Mason, and Joel D. Boerckel, et al. "Bone marrow mechanotransduction in porcine explants alters kinase activation and enhances trabecular bone formation in the absence of osteocyte signaling." Bone 107 (2018): 78-87.

How to cite this article: Jenneke Klein. "Signalling by osteocyte cells in relation to bone." *J Mol Hist Med Phys*. 7 (2022): 30.